Needle versus Tube Thoracostomy in a Swine Model of Traumatic Tension Hemopneumothorax

John B. Holcomb, MD, John G. McManus, MD, MCR, S. T. Kerr, MD, Anthony E. Pusateri, PhD

Abstract

Objective. Traumatic tension hemopneumothorax is fatal if not treated rapidly. However, whether prehospital decompression is better achieved by chest tube or needle thoracostomy is unknown. We conducted this study to compare the immediate results and prolonged effectiveness of two methods of treatment for traumatic tension hemopneumothorax in a swine model. **Methods.** Ten percent of calculated total blood volume was instilled into the hemithorax of spontaneously ventilating swine ($n = 5$ per group, 40 ± 3 kg). A Veres needle and insufflator were used to induce tension hemopneumothorax. Animals were randomized to one of four groups: 1) needle thoracostomy with 14-gauge intravenous catheter; 2) needle thoracostomy with Cook catheter; 3) 32-F chest tube thoracostomy; or 4) no intervention (control). Serial chest x-rays were obtained to document mediastinal shift before and after treatment. Arterial blood gas values and physiologic data were recorded. Postoperatively, thoracoscopy was performed to detect possible pulmonary injury from the procedure and/or catheter kinking or clotting.**Results.** Positive intrapleural pressure was rapidly relieved in all treated animals. Four-hour survival was 100% in the 14-gauge needle and chest tube thoracostomy groups, 60% in the Cook catheter group, and 0% in the control animals ($p < 0.05$). There were no significant differences in survival or physiologic measurements among the treated animals ($p > 0.05$). **Conclusions.** In this animal model, needle thoracostomy using a 14-gauge or Cook catheter was as successful as chest tube thoracostomy for relieving tension hemopneumothorax. **Key words:** tension pneumothorax; tube thoracostomy; needle thoracostomyn

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Introduction

If untreated, tension hemopneumothorax (THP) often becomes a fatal consequence of chest trauma. For combat casualties, THP is the second leading cause of potentially preventable prehospital death and the third leading cause of combat mortality overall.[1] To achieve chest decompression, the Advanced Trauma Life Support (ATLS) training manual recommends needle thoracostomy (NT) followed by chest tube thoracostomy (CTT).[2] The military version of the Prehospital Trauma Life Support (PHTLS) training manual currently recommends NT.[3] Based on current combat epidemiology, basic U.S. Army prehospital medical personnel ("91Whiskeys") are now routinely trained to perform NT using a 14-gauge 3.25-inch intravenous catheter during their initial schooling and certification. 91Whiskeys are only shown CTT and are not trained with hands-on experience. Thus, in the military environment with multiple casualties, few providers, and prolonged casualty evacuation times, significant time may elapse before conversion of NT to CTT.

Prospective comparison of NT and CTT to relieve THP in human trials has not been performed. However, a few retrospective studies in the prehospital arena have reported data on the efficacy of NT and CTT for THP.[4–6] Also, several studies have shown that NT alone may be insufficient to relieve a THP.[7–13] Currently, the correct initial technique and the safe time span between the two procedures are unknown. Delineating the fastest, simplest, yet most effective technique to safely restore negative intrapleural pressure (IP) is critical for remedying THP and reducing morbidity and mortality rates following traumatic chest injuries in both the combat and civilian trauma settings.[4,14–16]

The objectives of this study were: 1) to compare the initial efficacy of two different types of chest decompression procedures and 2) to document the continued efficacy of these treatment modalities in an animal model. We hypothesized that NT or CTT would relieve initial tension physiology with equal effectiveness and would continue to maintain negative IP for a minimum of four hours.

Methods

Animal Model

The Animal Care and Use Committee of the Institute of Surgical Research, Ft. Sam Houston, Texas, approved

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the protocol for this study. All animals received humane care in strict compliance with the *Guide for the Care and Use of Laboratory Animals* (National Institutes of Health publications 86-23, revised 1996). Twenty-four crossbred commercial swine weighing 40 ± 3 kg were used in this study. Groups of four to eight animals were housed indoors in enclosed runs. The pigs were fed a complete corn–soybean meal–based ration of 2.0 kg/pig/day, and water was available ad libitum. All animals were maintained in a facility accredited by the Association for Assessment and Accreditation of Laboratory Animal Care International.

Surgical Procedure

Animals were fasted 24 hours prior to surgical procedures, with water allowed ad libitum. Preoperative anesthesia was induced by masking the pigs with isofluorane in medical air, followed by ketamine (2.2 mg/kg) and atropine (0.05 mg/kg) injected intramuscularly. The pigs were then intubated with a 7.5-mm intradiameter cuffed endotracheal tube. Continuous isofluorane (1–1.5%) maintained surgical anesthesia. Injections of an equal mixture of 10 mL of 1% lidocaine and 0.5% marcaine were administered at all operative sites to control incisional pain and thereby decrease the requirement for isofluorane. Positive end-expiratory pressure was set at 3 mmH_2O, and the animals were kept in a state of spontaneous respiration on medical air (fraction of inspired oxygen [FiO_2] = 0.21). Intramuscular ketamine injections (1.1 mg/kg) were repeated every 75 minutes. This anesthesia regimen permitted spontaneous ventilation and capture of the animals' respiratory parameters through a closed system.

Following induction of anesthesia, vascular catheters were inserted into the right internal jugular vein, right carotid artery, and left and right femoral arteries. Lactated Ringer's solution was infused as a maintenance fluid at a rate of 1.0 mL/kg/h via the right internal jugular venous line. For the entire study the carotid arterial catheter was connected to a continuous blood pressure/waveform analyzer (Micro-Med, Louisville, KY) to record systolic blood pressure, diastolic blood pressure, mean arterial pressure (MAP), and heart rate every 10 seconds, averaged from readings taken every 2 seconds. Additionally, a fiberoptic continuous cardiac output and mixed venous Swan-Ganz catheter was placed through the venous line (OptiQ 8F and Oximetrix 3 SO2/CO computer and Qvue monitor, Abbott Critical Care Systems, Mountain View, CA). After the Swan-Ganz catheter was positioned, its location was documented with intraoperative fluoroscopy. All further instrumentation, creation of THP, and interventions were performed in the side opposite the location of the Swan-Ganz catheter tip. Temperature and mixed venous saturation levels (SvO_2) were recorded by the Swan-Ganz catheter every 10 seconds. The cardiac output was updated every 30 seconds and recorded every 5 minutes. An arterial blood gas catheter (Paratrend 7 Continuous ABG Monitoring Catheter, Diametrics Medical, Inc., St. Paul, MN) placed through the right femoral arterial line took measurements every 10 seconds. The left femoral arterial line was used for taking blood samples.

Creation of Traumatic Tension Hemopneumothorax

The animal model was developed during a test phase to establish a reproducible THP. A Veres needle was inserted in the 6th intercostal space into an anterior axillary line. Ten percent of the animal's total estimated blood volume (EBV: 296 ± 13 mL) was withdrawn from the left femoral arterial line over 2 minutes and injected into the chest via the Veres needle. A standard laparoscopic insufflator modified with a precise airflow regulator was then connected to the Veres needle, allowing accurate insufflation flow volumes and continuous IPs to be recorded.

During development of the animal model, we found that an insufflation rate of 3 mL/kg/min yielded a fatal THP in 40-kg pigs within 20 minutes. The total lung capacity of each animal's hemithorax was determined to be approximately 55 mL/kg/hemithorax, or 2.2 L in a 40-kg animal.[17] These parameters were recorded during our animal model development and were correlated with serial intraoperative chest x-rays to detect movement of the mediastinum. THP was defined by a mean positive IP greater than 1 mm Hg and significant deviation from baseline values of a least three other parameters (Table 1).

Treatment Groups

Twenty animals were divided into four groups of five animals each. Animals were assigned randomly to treatment by weight using a random numbers table[18] to achieve randomization while controlling for potential effects of body weight. Group 1 animals underwent NT with a 14-gauge, $2^1/_4$-inch intravenous catheter. Group 2 animals underwent NT with a Cook pneumothorax

TABLE 1. Requirements for Creation of Tension Hemopneumothorax

1.	Decrease in tidal volume (TV), 15% from baseline
2.	Decrease in cardiac output (CO), 20% from baseline
3.	Decrease in mixed venous oxygenation (SvO_2), 25% from baseline
4.	Increase in pulmonary artery pressure (PAP), 30% from baseline
5.	Intrapleural pressure (IPP) greater than 1 mmHg throughout the respiratory cycle

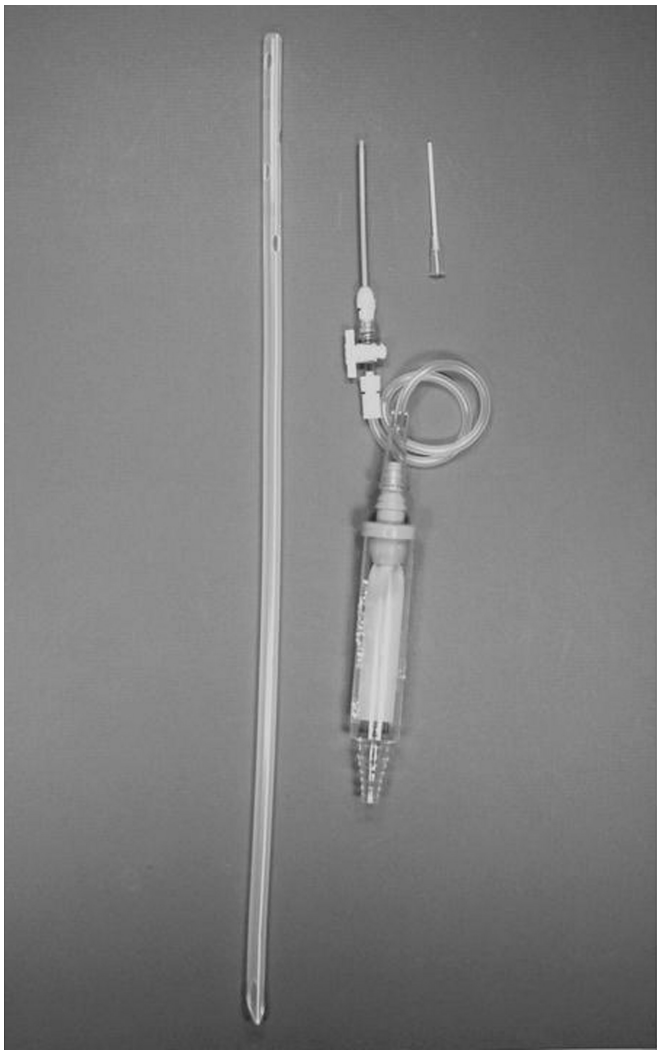

FIGURE 1. Photograph of (*left to right*) a 32-Fr chest tube, a 15-gauge Cook catheter, and a 14-gauge intravenous catheter.

set (Cook Critical Care, Bloomington, IN). The kit contains an 8.5-Fr by 7.5-cm straight reinforced catheter with a 15-gauge needle and a Heimlich valve (part number, C-TPTS-8.5-7.5-FSNS). Group 3 animals underwent CTT for which we used a 32-Fr Argyle thoracic catheter (Sherwood Medical, St. Louis, MO). Group 4 control animals received no intervention. All treatment catheters (Fig. 1) were inserted into the 6th intercostal space midaxillary line. A rush of air was heard in every case, with no adjustment made beyond initial attempt. Chest tubes were connected directly to a Heimlich valve, and the thoracostomy needles were connected to a Heimlich valve via a 12-cm length of latex tubing.

All catheters used in this study discharged pleural air at a rate of up to 3 L/min without an increase in IP, confirming that increased IP was not due to inherent resistance from small-caliber catheters. Insufflation at 3 mL/kg/min continued during and after treatment. The study plan called for no additional treatment if the original treatment failed and the animal redeveloped THP; however, no animals redeveloped THP.

Outcome Measurements

The following physiologic variables were monitored beginning 10 minutes prior to creation of the THP and continued for four hours after injury, or until death, whichever occurred first: core temperature, heart rate, systolic and diastolic blood pressures, MAP, continuous cardiac output, continuous mixed venous saturations, central venous pressure (CVP), pulmonary artery pressure (PAP), IP and peak airway pressures, respiratory rate, and tidal volume. Arterial blood gas (ABG) values were recorded immediately prior to Veres needle insertion, every 6 minutes during insufflation, 15 seconds prior to intervention, 1 minute after intervention, at 6-minute intervals for the first 30 minutes after intervention, and then at 60-minute intervals over the next four hours, or at death if it occurred prior to the planned study conclusion. All noncontinuous variables were also evaluated at the same time points.

The location of the mediastinum was monitored by chest x-ray (Fig. 2). Prior to insufflation, a radio-opaque J-wire was inserted into the right external jugular vein, through the right atrium, and into the inferior vena cava. Serial digital C-arm chest x-rays allowed measurement of the shift from midline of the wire to document any shifts in the mediastinum that resulted from THP formation and treatment. Chest x-rays were obtained every 3 minutes during insufflation, 15 seconds prior to intervention, 1 minute after intervention, and at the same time points as the blood samples were taken.

At study conclusion, all thoracostomy catheters were evaluated for intraluminal obstruction by flushing saline through the catheters and recording the presence or absence of clots. Thoracoscopic evaluation was performed in all animals to evaluate chest cavities for unintended pulmonary injury, or kinking of the thoracostomy catheters.

Death was confirmed when the MAP was less than 15 mmHg and the heart rate was 0. After the 240-minute study period, each surviving animal was euthanized with a concentrated solution of sodium pentobarbital.

Statistical Analysis

Data were analyzed using SAS statistical software (version 8.1, SAS Institute, Cary, NC). Survival times were compared among the four groups using the log-rank test. Continuous measurements taken over ten different time points were analyzed using a hierarchical mixed-model analysis of variance (ANOVA). These time points were baseline, insufflation, 0 for the intervention time point, and 5, 10, 30, 60, 120, 180, and 240 for the times in minutes after the intervention. Values used for analysis were measured at all time points except

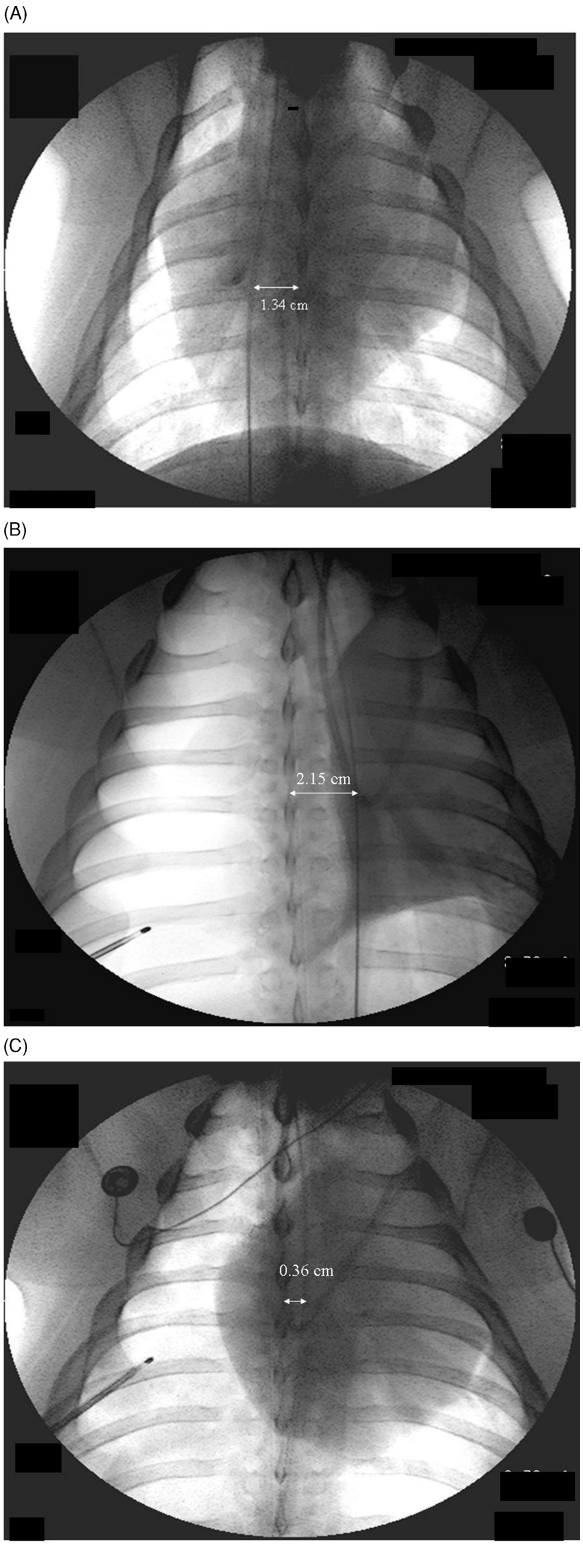

FIGURE 2. Chest radiographs demonstrating creation and treatment of tension hemopneumothorax. **A**: Preinsufflation baseline: J-wire in the inferior vena cava 1.34 cm to the right of the spinous processes. **B**: Insufflation resulted in a mediastinal shift of 2.15 cm to the left of the spinous processes, resulting in a total shift of 3.49 cm from baseline. **C**: Partial return of mediastinum immediately after treatment with a needle catheter.

insufflation. Values for insufflation, which took place over a 5-minute interval, were averaged. The treatment, time, and treatment-by-time interaction were considered fixed effects. Data were analyzed by repeated-measures ANOVA using a random subject effect. Comparisons of measurements between successive time points throughout the experiment and comparisons of measurements at successive time points with baseline were analyzed using one-way ANOVA and paired t-test. p-Values of multiple analyses and comparisons were adjusted using Hochberg's (1988) step-up Bonferroni method. Statistical significance was noted at $p < 0.05$.

RESULTS

All animals exhibited similar preinsufflation and preintervention physiologic variables, with a mean (± standard error of the mean [SEM]) time to intervention of 384 ± 41 seconds. No evidence of catheter kinking or clotting or pulmonary injury was noted at study conclusion. There were significant differences in survival time between the control group and each of the three treatment groups ($p = 0.0018$ for each comparison), but comparisons between treatment groups revealed no significant differences ($p = 0.9$). The Kaplan-Meier plot displays estimates of survival functions for all groups in the study (Fig. 3). Survival rates were 0% for the control group, 60% for the Cook NT group, 100% for the 14-gauge NT group, and 100% for the CTT group. The mean survival time for the control group was 12.6 ± 3.6 minutes. In the Cook NT group, survival times were 45 and 48 minutes for two animals, whereas the other three animals in this group survived the four-hour

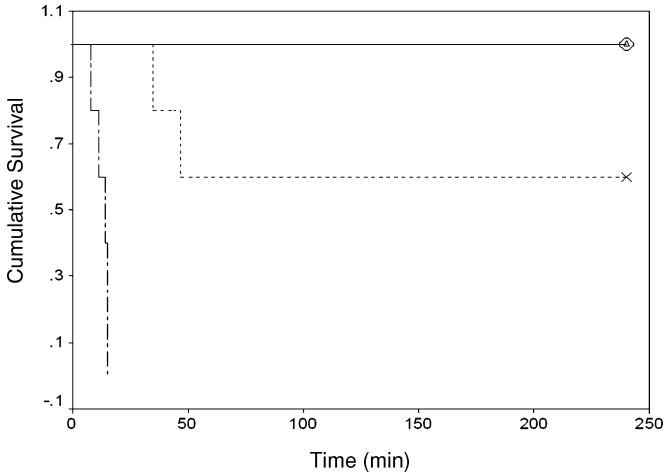

FIGURE 3. Kaplan-Meier plot displaying estimates of the survival functions for all study groups. Survival rates for both the chest tube thoracostomy and 14-gauge needle thoracostomy groups were 100%. Survival for the Cook needle thoracostomy group was 60%.

study period. In the CTT and 14-gauge NT groups, all animals survived the four-hour study period.

Table 2 summarizes the mean physiologic data and the mean x-ray data. For the sake of brevity, time points at 30 and 180 minutes were omitted because they represented insignificant physiologic data. X-ray data were calculated from the shift of the mediastinum following insufflation and measured in centimeters. Figure 4 displays the profiles of the physiologic and x-ray variables for the four groups over the successive time points from baseline to the end of the study.

Comparisons among Treatments

There were no significant differences among treatment groups in profiles of the physiologic and x-ray

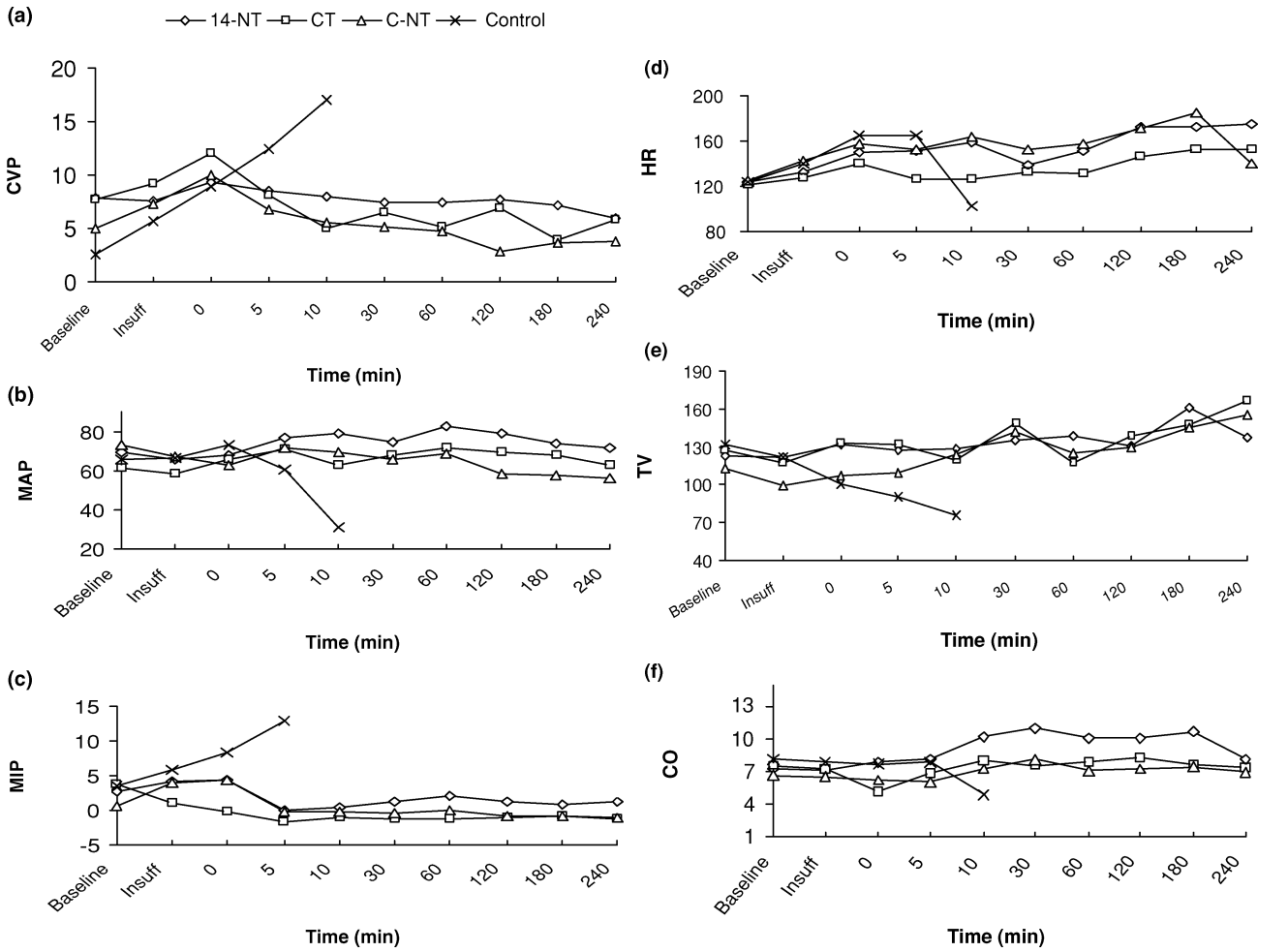

FIGURE 4. Profiles of all groups for central venous pressure (CVP), mean arterial pressure (MAP), mean intrapleural pressure (MIP), heart rate (HR), tidal volume (TV), cardiac output (CO), mixed venous oxygenation (SvO_2), pulse oximetry pressure (SpO_2), end-tidal carbon dioxide ($ETCO_2$), pH, and mediastinal shift (MedS). Insuff = insufflation. **A**: CVP increased similarly during insufflation for all groups, and then diverged at 5 minutes after intervention ($p < 0.0001$), with CVP increasing further in control animals while approaching baseline after intervention and remaining stable throughout the remainder of the study for all treatment groups. **B**: MAP was stable during insufflation for all groups, but diverged at 10 minutes after intervention ($p < 0.0001$), decreasing in the control group but remaining stable in the treatment group. **C**: MIP increased during insufflation in all groups, and then diverged at 5 minutes after intervention ($p = 0.015$), with MIP continuing to increase in the control group and decreasing below baseline in the treatment groups, where it remained for the remainder of the study. **D**: HR increased during insufflation for all, and then diverged at 10 minutes after intervention ($p = 0.0052$), with HR dropping in the control group and remaining elevated in the treatment groups. **E**: TV was stable and close to baseline during insufflation for all, but tended to diverge at intervention ($p = 0.054$), with TV dropping in the control group and remaining stable in the treatment group. **F**: The CO seemed stable in all groups during insufflation, and then CO in control tended to decrease at 10 minutes compared with baseline. Postintervention CO remained mostly stable in the treatment groups throughout the study. **G**: SvO_2 decreased during insufflation, and then increased for all groups from 5 minutes to 10 minutes after treatment. For the treatment groups, SvO_2 approached baseline within 5 minutes after treatment and remained stable throughout the study. **H**: SpO_2 decreased during insufflation for all groups, and then remained stable for all groups from 5 minutes to 10 minutes after treatment. For the treatment groups, SpO_2 continued to remain stable throughout the study without returning to baseline. **I**: $ETCO_2$ was stable for all treatment groups during the study, but became elevated about 5 minutes before death in the control group. **J**: pH seemed stable in all treatment groups, but decreased about 10 minutes before death in the control group. **K**: MedS increased similarly during insufflation for all, and then diverged at 10 minutes after intervention ($p = 0.002$), with MedS still increasing in the control group while approaching a return to baseline in the treatment group. *(Continued)*

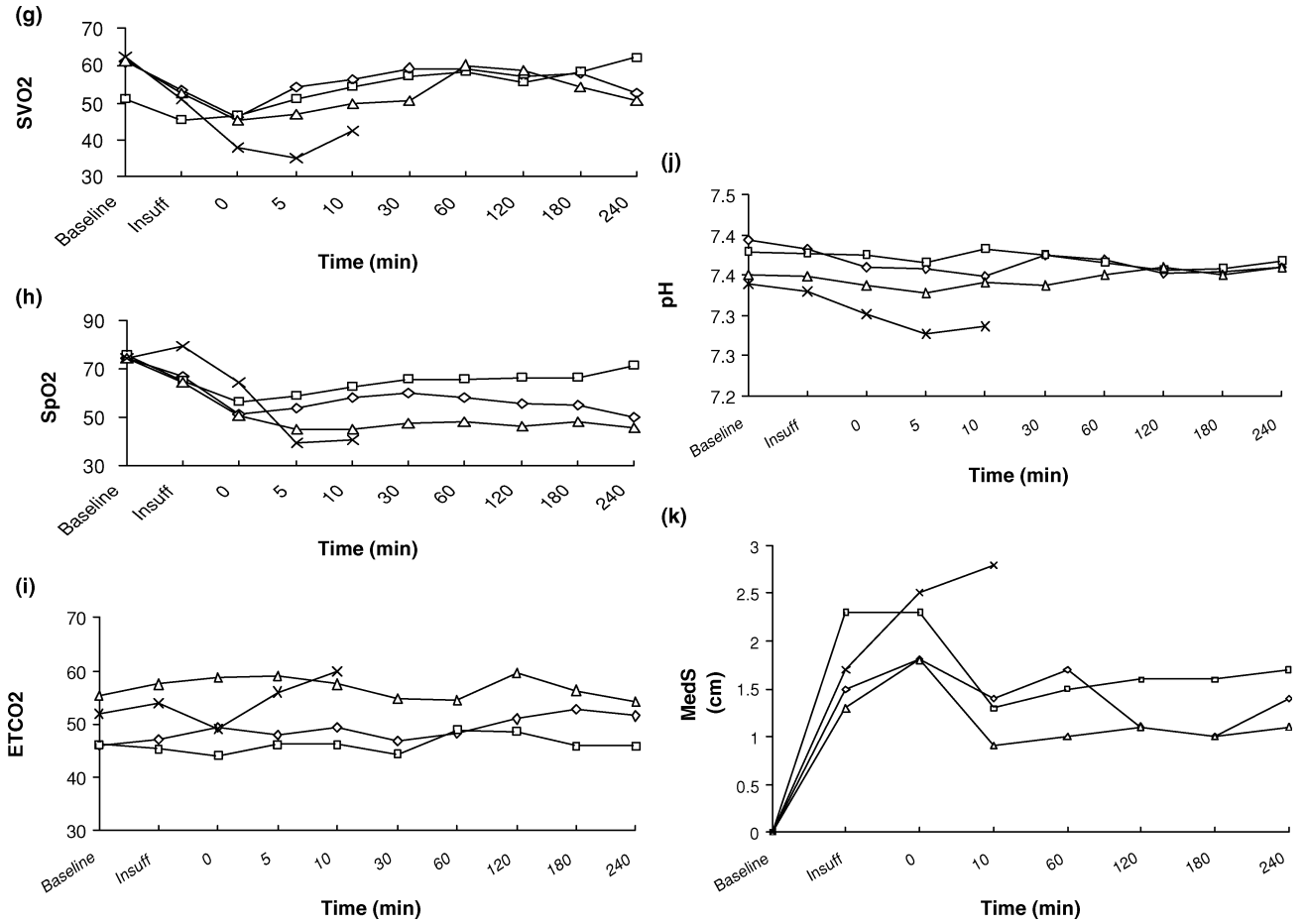

FIGURE 4. (Continued)

parameters ($p \geq 0.92$ for all). Also, there were no significant differences among treatment groups in the physiologic and x-ray parameters averaged over time ($p = 0.99$ for all).

Comparisons among Treatments and Control

Normal IP was restored within 60 seconds after intervention, and CVP returned to baseline within 180 seconds. The profile for the control group was significantly different from that of each treatment in CVP ($p \leq 0.003$), mean intrapleural pressure (MIP) ($p \leq 0.003$), MAP ($p \leq 0.003$), heart rate ($p < 0.007$), tidal volume ($0.0012 < p < 0.04$), and x-ray parameter (mediastinum) ($p < 0.014$) from baseline to 10 minutes after intervention (i.e., the time point before death in control).

No significant differences in profiles among the four groups were found in pulse oximetry pressure (SpO_2) ($p = 0.55$), SvO_2 ($p = 0.55$), cardiac output (CO) ($p = 0.11$), pH ($p = 0.55$), and end-tidal carbon dioxide ($ETCO_2$) ($p = 0.55$) from baseline to 10 minutes after intervention.

DISCUSSION

Records of the seriousness of chest trauma have been available since the 16th century, when medical practitioners stated that virtually all penetrating wounds to the thorax were untreatable and fatal.[14] With respect to combat injuries, autopsy studies from the Vietnam War strongly suggested that untreated isolated tension pneumothorax led to between 3% and 5% of all cases of potentially preventable prehospital deaths (Fig. 5).[1] The mortality rates of different conflicts—62.5% in the Civil War, 74% in World War I, and 61% in World War II—emphasize the significance of chest injury on the battlefield.[19] Thus, two factors in combat trauma are considered primarily responsible for preventable deaths from THP: 1) failure to diagnose and 2) lack of an appropriate early intervention in the far forward environment.

Significant morbidity and mortality from THPs are also present in civilian trauma, where chest injuries account for approximately one-fourth of all trauma deaths and are a contributing factor in 50% of remaining fatalities.[4] However, despite these statistics, more than 90% of all chest trauma can be resolved with airway management, fluid resuscitation, and chest

TABLE 2. Mean Physiologic Data for the Study Time Points

Time	CVP Control	Trt*	MAP Control	Trt	MIP Control	Trt	SpO_2 Control	Trt	TV Control	Trt	HR Control	Trt	$ETCO_2$ Control	Trt	MedS Control	Trt
Baseline (±SD)	2.6 (2.6)	6.8 (7.3)	65.8 (9.7)	68.0 (13.5)	3.6 (1.9)	2.4 (3.3)	74.3 (18.8)	74.7 (14.7)	132.0 (20.5)	121.0 (28.5)	123.7 (17.1)	123.0 (20.5)	51.7 (6.1)	49.1 (8.8)	0.87 (0.54)	0.48 (0.40)
Insufflation (±SD)	**5.6** (3.1)	**9.1** (7.3)	66.7 (10.3)	64.1 (17.9)	**6.0** (2.7)	3.3 (3.1)	**79.3** (41.0)	**66.2** (12.2)	121.4 (22.1)	113.0 (26.0)	**140.0** (32.7)	**133.4** (29.2)	53.9 (6.8)	49.9 (9.3)	0.85 (0.58)	1.26 (0.56)
0 min (±SD)	**9.0** (2.4)	10.4 (7.5)	73.1 (16.3)	65.5 (17.9)	8.3 (5.0)	3.1 (3.2)	**64.2** (54.5)	**52.3** (10.1)	**100.0** (16.8)	123.2 (47.5)	**164.6** (26.0)	**149.9** (36.6)	49.0 (7.6)	51.2 (10.0)	1.60 (0.99)	1.50 (0.36)
5 min (±SD)	**13.0** (2.2)	8.1 (7.1)	59.8 (18.1)	73.1 (16.2)	**12.8** (1.1)	**−0.6** (2.5)	**45.2** (14.7)	**53.8** (12.2)	**90.8** (21.7)	123.0 (40.4)	**171.6** (38.2)	**142.9** (33.2)	56.1 (5.7)	51.0 (10.7)	NA	NA
10 min (±SD)	**17.1** (1.3)	5.8 (4.8)	**30.0** (9.5)	72.7 (14.6)		**0.2** (2.8)	**40.5** (10.0)	**54.7** (12.1)	**76.3** (23.0)	123.8 (35.4)	**90.1** (65.7)	**153.4** (37.1)	59.7 (2.1)	51.0 (10.5)	1.92 (0.77)	0.67 (0.37)
60 min (±SD)		6.0 (4.6)		75.0 (10.9)		0.4 (3.9)		**58.7** (12.4)		127.5 (38.5)		**145.3** (47.3)		49.9 (6.4)		0.82 (0.40)
120 min (±SD)		6.1 (5.5)		70.5 (15.7)		**−0.1** (3.4)		**57.7** (13.9)		133.8 (30.6)		**162.1** (34.3)		51.3 (7.7)		0.72 (0.59)
240 min (±SD)		5.4 (3.8)		65.2 (9.6)		**−0.2** (3.7)		**58.7** (15.5)		**152.1** (32.1)		**159.9** (28.5)		49.6 (6.9)		

Boldface values denote significant change from baseline within group at $p < 0.05$.

*Average of all three treatment groups.

CVP = central venous pressure; $ETCO_2$ = end-tidal carbon dioxide; HR = heart rate; MAP = mean arterial pressure; MedS = measurement of mediastinal shift from baseline (cm); MIP = mean intrathoracic pressure; NA = not applicable; SD = standard deviation; SpO_2 = pulse oximetry pressure; Trt = treatment; TV = tidal volume.

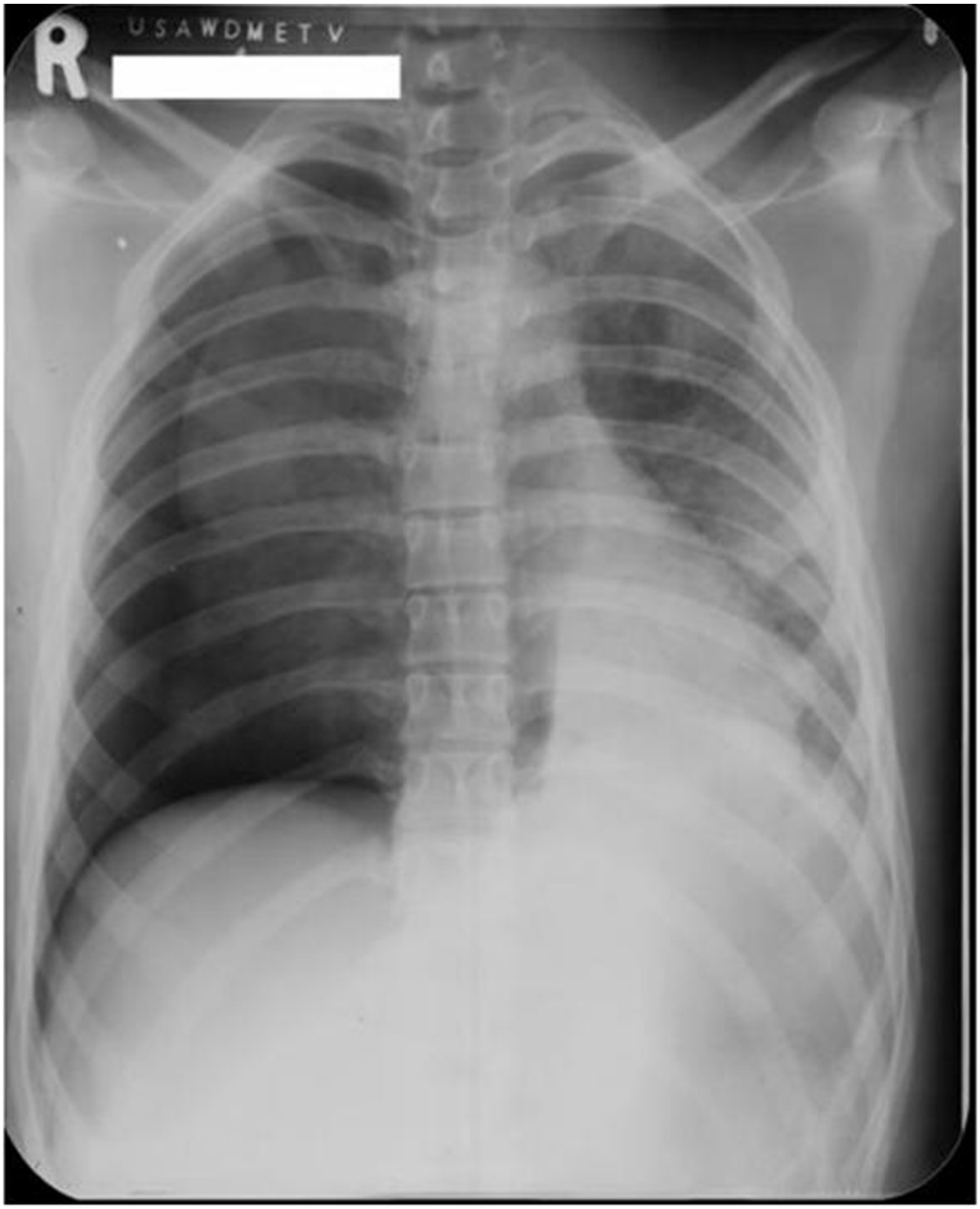

FIGURE 5. Postmortem chest radiograph of a soldier in the Vietnam War who died of an isolated tension pneumothorax. Note the mediastinal shift similar to animal model insufflation (Fig. 2B).

decompression.[20] The actual incidence of THP in the civilian prehospital setting varies with the specific population studied and has ranged from 0.7% to 30%.[21]

Whereas other modalities, such as the McSwain dart, have been used in the past, their popularity has rapidly decreased as a result of potential (never proven) pulmonary parenchymal injury and product liability issues.[22,23] The practice of CTT in both the civilian and combat prehospital settings remains controversial.[4,21] Some literature has reported the ability of advanced prehospital providers to successfully relieve THP with minimal complications and lower mortality as compared with use of NT alone.[4] The use of NT has also been shown to be effective and safe in the prehospital setting.[6,15] However, several retrospective studies have reported NT to be inadequate in certain cases as well as often overzealously used.[7–13] Despite some of these problems associated with the use of prehospital NT, it is currently the procedure of choice, for the majority of prehospital personnel, to treat THP.[2,3]

In the current study, we compared the efficacy of two different types of NT with standard CTT for the treatment of THP in a swine model. Furthermore, we wanted to determine not only whether NT has the potential to be an effective initial therapeutic intervention for THP, but also whether it will continue to maintain

function without resulting in a continued air leak, or fail because of kinking or clotting of the catheter, as previously reported in the literature.[7–13]

In our experiments at the time of intervention, MIP, CVP, and PAP were elevated compared with baseline, thereby supporting the need for immediate treatment. However, although many variables (CO, MAP, etc.) did not drastically change once THP was created, injury severity was proven by the fact that all five control animals died within 20 minutes after insult. Survival was 100% in the 14-gauge NT and CTT groups and 60.0% in the Cook NT group. Although these differences in survival time among the three treatments were not statistically significant ($p = 0.9$), our data suggest that NT was as effective as CTT in treating THPs. There was no difference among the physiologic parameters for the three treatments over the successive time points from baseline to the end of the study ($p = 0.92$). Likewise, the mediastinal shift was similar among all treatments. All animals underwent thoracoscopy to evaluate for parenchymal damage and catheter kinking. In the animals that survived the study period, thoracoscopy was completed prior to the animals' euthanasia. No evidence of significant catheter kinking or clotting or pulmonary injury in any group was noted at study conclusion, thereby supporting the safety of these procedures in this model.

As mentioned previously, reports of clinical cases cite several problems with NT, such as insufficient thorax penetration, intercostal artery and great vessel laceration, and pulmonary parenchymal injury.[7–13,21,24] Length of needle and anatomic location of needle placement have attempted to reduce failed NT for THP.[11,22] Certainly these problems will have to be addressed in the future application of prehospital NT for THP. However, needle thoracostomy with a long (5¼-inch) 14-gauge intravenous catheter seems to be an achievable training objective as it is fundamentally fast and simple, and poses minimal logistic and financial burdens. Currently, the U.S. Army medics carry 14-gauge intravenous catheters to treat tension pneumothoraxes. Also, many civilian agencies supply their prehospital personnel with the Cook NT catheter, which is specifically designed for relieving a tension pneumothorax. In general only advanced prehospital providers (combat paramedics, physician assistants, nurses, etc.) in both the civilian and military settings possess the minimal equipment and/or expertise required to perform CTT.

Limitations

This study has a few limitations worthy of discussion. First, our study was performed in a controlled environment with good lighting and with the diagnosis known beforehand. We acknowledge that in a prehospital trauma setting, standard physical examination techniques to diagnose a THP such as auscultation, palpation, and observation are difficult, if not impossible, as documented in previous literature.[4] Considering the likelihood of death for an untreated tension pneumothorax, the presence of respiratory difficulty coupled with the presence of a penetrating injury may have to suffice as the indications for thoracostomy on the battlefield. These are the current indications for NT in the Tactical Combat Casualty Care training given to all Department of Defense medics. Also, our procedure used to create a THP in swine may not be indicative of the traumatic injuries seen in "real-world" settings. Many of the current explosive injuries create multiple thoracic injuries, which may not respond as a single injury as in our model. Finally, our study has not been prospectively validated in human subjects.

CONCLUSION

The development of a traumatic THP is a well-described clinical entity with a documented rapidly fatal outcome if not treated successfully. Immediate successful treatment may consist of either needle or chest tube decompression. In this study we have demonstrated that needle decompression, when done properly, over a four-hour period is as effective as traditional chest tube decompression in an animal model. Needle decompression is a simple, fast, and effective method for treatment of THP and should be continued to be taught to prehospital providers treating traumatic chest injuries.

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